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The V_2 Transition Ratio

A New Electrocardiographic Criterion for Distinguishing Left From Right Ventricular Outflow Tract Tachycardia Origin

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Objectives We sought to develop electrocardiography (ECG) criteria for distinguishing left ventricular outflow tract (LVOT) from right ventricular outflow tract (RVOT) origin in patients with idiopathic outflow tract ventricular tachycardia (OTVT) and lead V_3 R/S transition.

Background Several ECG criteria have been proposed for differentiating left from right OTVT origin; ventricular tachycardias (VTs) with left bundle branch block and V_3 transition remain a challenge.

Methods We analyzed the surface ECG pattern of patients with OTVT with a precordial transition in lead V_3 who underwent successful catheter ablation. Sinus and VT QRS morphologies were measured in limb and precordial leads with electronic calipers. The V_2 and V_3 transition ratios were calculated by computing the percentage R-wave during VT $(R/R+S)_{VT}$ divided by the percentage R-wave in sinus rhythm $(R/R+S)_{SR}$.

Results We retrospectively analyzed ECGs from 40 patients (mean age 44 ± 14 years, 21 female) with outflow tract premature ventricular contractions (PVCs)/VT. Patients with structural heart disease, paced rhythms, and bundle branch block during sinus rhythm were excluded. The V_2 transition ratio was significantly greater for LVOT PVCs compared with RVOT PVCs (1.27 ± 0.60 vs. 0.23 ± 0.16; $p < 0.001$) and was the only independent predictor of LVOT origin. In 21 prospective cases, a V_2 transition ratio ≥0.60 predicted an LVOT origin with 91% accuracy. A PVC precordial transition occurring later than the sinus rhythm transition excluded an LVOT origin with 100% accuracy.

Conclusions The V_2 transition ratio is a novel electrocardiographic measure that reliably distinguishes LVOT from RVOT origin in patients with lead V_3 precordial transition. This measure might be useful for counseling patients and planning an ablation strategy. (J Am Coll Cardiol 2011;57:2255–62) © 2011 by the American College of Cardiology Foundation

Outflow tract ventricular tachycardia (OTVT) represents the most common subgroup of idiopathic ventricular tachycardia (VT). It typically occurs in healthy patients of young to middle age without structural heart disease and might be provoked by emotional stress, exercise, or dietary stimulants (1). The clinical presentation of OTVT is heterogeneous, ranging from isolated premature ventricular contractions (PVCs) to repetitive nonsustained VT to sustained VT. Although this diagnosis can be malignant, it generally carries an excellent prognosis and can be effectively treated by drugs or radiofrequency (RF) catheter ablation (2–5).

Detailed intracardiac electrical mapping has demonstrated that the vast majority of OTVTs originate from the anterior and superior septal aspect of the right ventricular outflow tract (RVOT), just inferior to the pulmonic valve (6,7). Less commonly, the site of origin can be localized to the right ventricular (RV) infundibulum, RV free wall, and posterior aspect of the interventricular septum. In approximately 10% to 15% of cases, the arrhythmia originates from the left ventricular outflow tract (LVOT) and can be mapped to the region of the aortic cusps (1,8–10). Rarely, OTVTs can be ablated from within the anterior interventricular vein, aorto-mitral continuity, or the root of the pulmonary artery.

Because OTVT has a focal origin and occurs in patients with structurally normal hearts, it is an arrhythmia that is

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Abbreviations and Acronyms

ECG = electrocardiogram/electrocardiography
LBBB = left bundle branch block
LCC = left coronary cusp
LVOT = left ventricular outflow tract
OT = outflow tract
OTVT = outflow tract ventricular tachycardia
PVC = premature ventricular contraction
RBBB = right bundle branch block
RCC = right coronary cusp
RF = radiofrequency
RV = right ventricle/ventricular
RVOT = right ventricular outflow tract
VT = ventricular tachycardia

particularly conducive to localization with the 12-lead electrocardiography (ECG). Typically, OTVT originating in the RV manifests an inferior axis in the frontal plane and left bundle branch block (LBBB) configuration with precordial R/S transition at or after lead V_3 (11–13). By contrast, LVOT VT usually manifests either a right bundle branch block (RBBB)/inferior axis or a LBBB/inferior axis with a precordial R/S-wave transition at or before lead V_3 (1,14). The ECG characteristics have also been described for idiopathic VT foci originating from the aorto-mitral continuity, anterior interventricular vein, and the left coronary cusp (LCC)–right coronary cusp (RCC) junction (6,10,11,13–15).

Criteria to distinguish RVOT from LVOT origin for patients with precordial transition occurring at lead V_3 have been lacking. This is of particular clinical importance, because a substantial number of OTVTs demonstrate lead V_3 transition and the need for left-sided ablation significantly alters patient counseling with regard to procedural time and risk (16). The aim of this study was to develop an ECG algorithm for reliably predicting the site of origin of OTVTs with lead V_3 precordial R/S transition. Existing ECG algorithms currently do not take into account cardiac rotation, respiratory variation, or the position of ECG leads on the chest, which might vary depending on body habitus, breast size, and technician expertise when placing leads. We hypothesized that comparison of the PVC/VT with the sinus rhythm (SR) QRS morphology would be an effective means of distinguishing LVOT from RVOT VT.

Methods

Study design. This study was designed in 2 parts: 1) a retrospective review of OTVT ablation cases in order to develop the ECG algorithm; and 2) a prospective assessment of the algorithm on a second group of patients.

Patient selection. We reviewed records on all patients who underwent mapping and ablation of idiopathic PVCs or VT at the Hospital of the University of Pennsylvania between January 2002 and December 2009. Patients with PVCs/VT manifesting a LBBB/inferior axis and a precordial transition (from R/S <1 to R/S >1) at lead V_3 were included. The majority of patients had normal left ventricular function by echocardiography, although patients with a presumed cardiomyopathy due to frequent ventricular ectopy were not excluded. Patients with ECG evidence of prior myocardial infarction, RBBB during SR, or whose clinical arrhythmia could not be abolished with catheter ablation were excluded.

Mapping and ablation protocol. A standard quadripolar catheter was positioned in the RV apical position, and a 4-mm nonirrigated catheter (Navistar or Celcius, Biosense-Webster, Diamond Bar, California) was initially positioned in the RVOT for mapping. In patients with sufficient ectopy, activation mapping was performed, recording the earliest local bipolar activation time compared with surface QRS of the clinical PVC. Pace-mapping at a threshold just above local capture was performed in all cases with careful comparison of the paced surface QRS morphology with that of the clinical PVC. All idiopathic PVCs/VT in this series originated from the septal side of the RVOT just beneath the pulmonic valve. The site of RVOT origin was classified by fluoroscopy and electroanatomic mapping as originating from either the anterior or posterior septal aspect of the RVOT for simplicity (12).

The decision to extend mapping to a LVOT site was made if no adequate RVOT sites were identified or ablation in the RVOT was unsuccessful in abolishing the arrhythmia. The LVOT sites were mapped via a retrograde aortic approach. All mapping was performed after heparin bolus, maintaining an activated clotting time >250 s. In addition to standard fluoroscopy, a 3-dimensional electroanatomic mapping system (CARTO, Biosense-Webster, or NavX, St. Jude Medical, Minnetonka, Minnesota) and intracardiac echocardiography (Acuson, Siemens Medical, Mountain View, California) were used to localize the anatomic position of the ablation catheter within the outflow tract (Fig. 1). For most of our experience, a nonirrigated 4-mm tip catheter with power delivered up to 50 W and temperature up to 50°C was used for RVOT and LVOT ablation. Beginning in 2008, irrigated 3.5-mm tip catheters were used almost exclusively in the LVOT and on occasion by some operators in the RVOT. In the RVOT, a maximum power setting of 30 to 35 W was used. For the LVOT/aortic cusp region, the typical maximal power setting was 30 W; however, in select cases with late termination of PVCs or suppression and recurrence of ectopy, powers of up to 50 W were used.

Acute ablation success was defined as the absence of the clinical PVC at 30 min after the last RF delivery, both with and without isoproterenol, and confirmed by continuous full disclosure cardiac telemetry in the subsequent 24 h of inpatient care.

ECG measurement protocol. Sinus rhythm and VT ECG morphology were measured on the same 12-lead ECG with electronic calipers on the Prucka CardioLab (GE Healthcare, Waukesha, Wisconsin) recording system. Standard 12-lead ECG electrode placement was used. Lead gain was uniform with paper speed of 100 mm/s. During the clinical arrhythmia, the following measurements were obtained during both SR and the PVC/VT: 1) R- and S-wave amplitudes in lead I, II, III, aVF, and V_1 to V_3; 2) R-wave

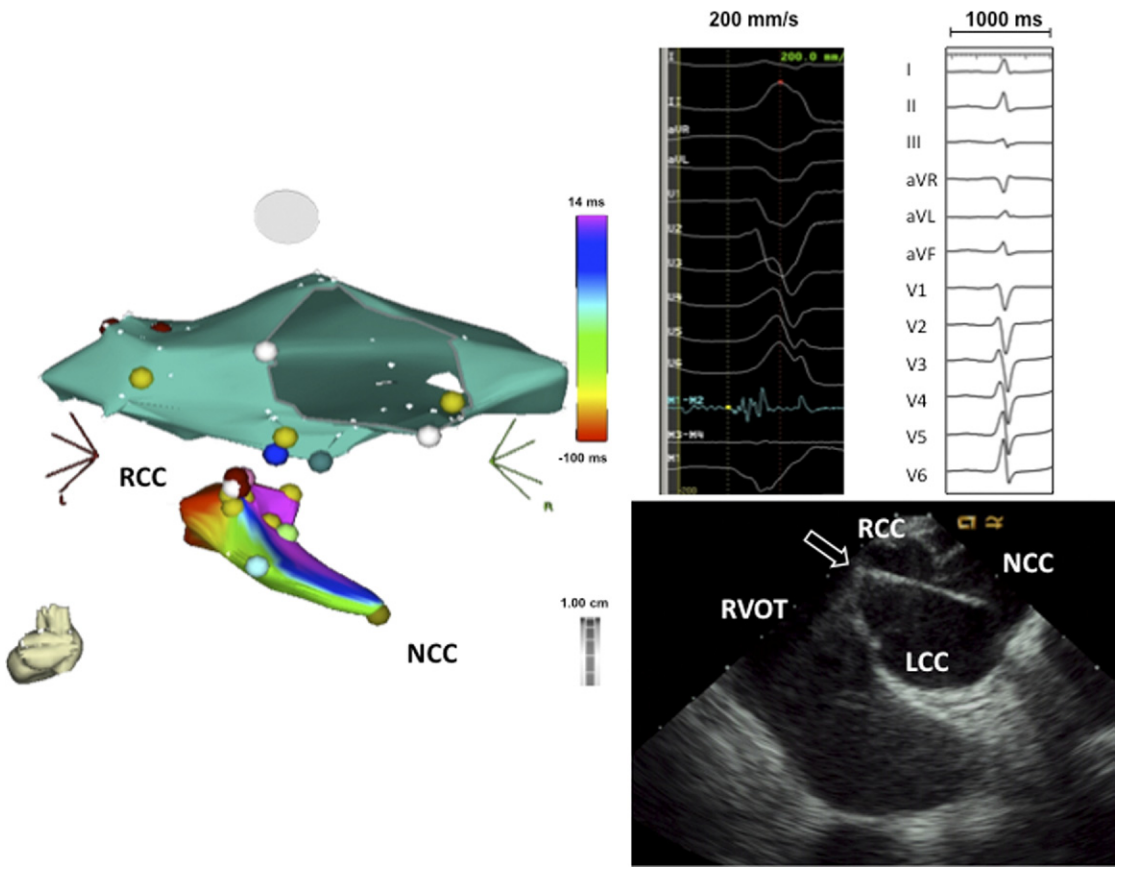

Figure 1 **Mapping PVC Origin During Catheter Ablation**

(Left panel) Electroanatomic map (superior view) of the right ventricular outflow tract (RVOT) and aortic cusps. The best pacemap in the posterior RVOT **(dark blue tag)** was not perfect, and activation was earlier in the right coronary cusp (RCC). Ablation at the right cusp **(red tag)** abolished the premature ventricular contraction (PVC). **(Upper right panels)** The PVC morphology is shown together with the bipolar intracardiac electrogram recorded at the site of earliest activation (M1-2), unipolar electrogram qS pattern (M1), and sinus rhythm electrocardiography morphology. Note that the precordial R-wave transition of the PVC is earlier compared with the sinus rhythm transition. **(Lower right panel)** The catheter location is shown in the RCC **(arrow)** with the intracardiac echo probe at the right ventricular infundibulum. The V_2 transition ratio in this patient was 0.62. LCC = left coronary cusp; NCC = noncoronary cusp.

duration in leads V_1 to V_3; 3) QRS duration; and 4) QRS frontal axis (Fig. 2). The T-P segment was considered the isoelectric baseline for measurement of R- and S-wave amplitudes. The QRS duration was measured from the site of earliest initial deflection from the isoelectric line in any lead to the time of latest activation in any lead. The R-wave duration was measured from the site of earliest initial deflection from the isoelectric line to the time at which the R-wave intersected the isoelectric line. For all cases, QRS measurements were performed on isolated PVCs representative of the clinical VT before the induction of sustained VT and compared with the SR QRS complex. As a means of accounting for respiratory variation, we standardized our SR measurements by measuring the largest R- and S-wave over a 10-s window at 25-mm/s sweep speed (Fig. 3). The transition ratio was calculated in leads V_2 and V_3 by computing the percentage R-wave during VT $(R/R+S)_{VT}$ divided by the percentage R-wave in SR $(R/R+S)_{SR}$.

We also assessed a simple, easily used, qualitative measure of comparing the precordial transition of the PVC/VT with SR. We hypothesized that a PVC/VT precordial transition to R>S at an interspace equal to or earlier than the location of the SR transition would suggest an LVOT origin, whereas a transition at an interspace later than the SR transition would suggest an RVOT origin. The sensitivity and specificity for this measure was assessed.

Prospective analysis. We then performed a prospective evaluation of a second cohort of consecutive patients with LBBB/inferior PVCs/VT with lead V_3 R/S transition who met the same inclusion criteria used in the retrospective cohort and who presented for catheter ablation between January 2010 and August 2010. Quantitative and qualitative measurements in this group were performed by 2 blinded observers, before any knowledge of the site of VT origin. The site of successful ablation was identified as the one that led to abolishment of the clinical PVC/VT. The utility of

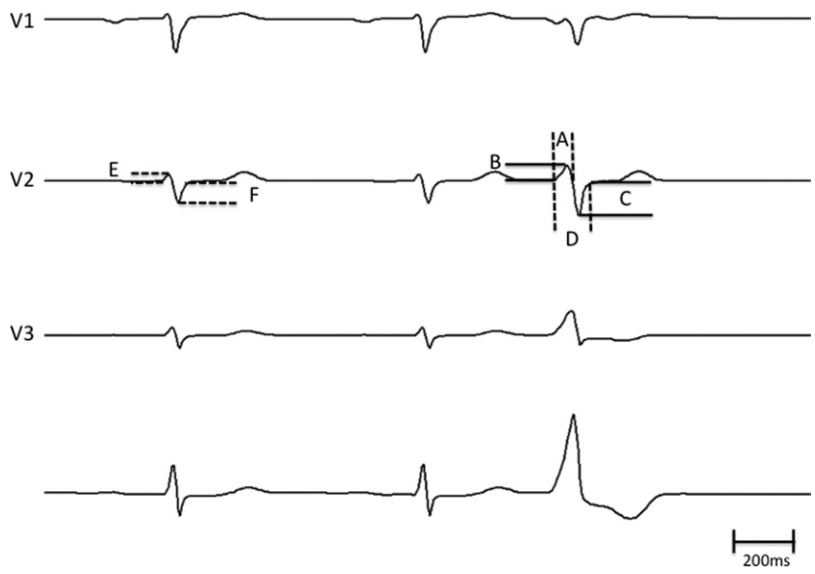

Figure 2 **Electrocardiographic Measurements**

Leads V_2 and V_3 of normal sinus beat followed by a premature ventricular contraction (PVC) representative of the clinical outflow tract ventricular tachycardia. Measurements are as follows: **A** = PVC R-wave duration (ms); **B** = PVC R-wave amplitude (mV); **C** = PVC S-wave amplitude (mV); **D** = PVC QRS duration (ms); **E** = sinus rhythm R-wave amplitude (mV); and **F** = sinus rhythm S-wave amplitude (mV). The transition ratio was calculated in each lead with the following formula: $[B/(B+C)_{VT} \div E/(E+F)_{SR}]$.

the precordial transition ratio for predicting the site of PVC/VT origin was then determined.

Statistical analysis. Continuous variables are presented as mean ± 1 SD. Continuous variables were compared with Student *t* test and categorical variables with Fisher exact test. Predictors with a p value <0.1 were entered into a stepwise forward multivariate logistic regression to determine independent predictors of PVC origin (LVOT vs. RVOT). Agreement between the ECG measurements of the blinded observers was assessed with the intraclass correlation coefficient, assuming a 2-way random model with absolute agreement. The significance of the intraclass correlation coefficient was assessed with an *F* test to determine whether the correlation was >0. The kappa statistic was also computed for agreement in classification between the 2 observers. A receiver operating characteristic curve was performed for sensitivity and specificity analysis. Statistics were performed on SPSS statistical software (release 18.0, SPSS, Chicago, Illinois). A p value ≤0.05 was considered statistically significant.

Results

Retrospective analysis. We reviewed data from 293 patients who underwent catheter ablation of OTVT between January 2002 and December 2009 at our institution. After excluding patients with precordial transition V_1 or V_2 (n = 49), precordial transition V_4 to V_6 (n = 35), structural or ischemic heart disease (n = 55), RBBB (n = 8), paced rhythm (n = 2), unable to acutely abolish PVC/VT (n = 5),

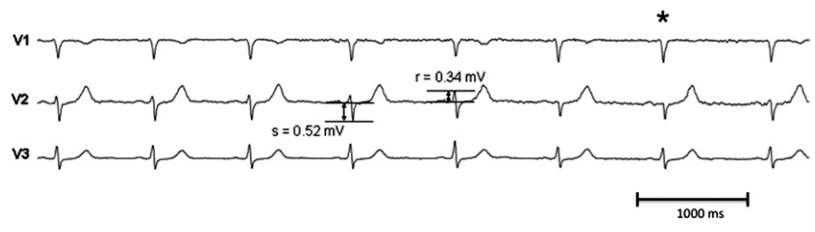

Figure 3 **SR ECG Tracing Demonstrating Respiratory Variation in R- and S-Wave Amplitude**

Surface electrocardiography (ECG) leads V_1 to V_3 recorded during sinus rhythm (SR) show marked respiratory variation of the R-wave in lead V_2. To standardize the measure, the largest R- and S-wave during a 10-s SR recording was used. The calculated V_2 transition ratio with the largest SR R- and S-wave amplitudes in this patient (labeled) was 0.34, correctly predicting right ventricular outflow tract origin. *If the seventh SR QRS complex alone was used, the V_2 transition ratio would be 3.60, incorrectly predicting left ventricular outflow tract origin.

Table 1 Baseline Demographic Data and Clinical Characteristics of the Retrospective Cohort

	Total (n = 40)	LVOT (n = 20)	RVOT (n = 20)	p Value
Age (yrs)	44.41 ± 14.29	47.70 ± 14.35	40.95 ± 13.74	0.142
Male (%)	48	65	40	0.205
Ethnicity (%)				
Caucasian	65	70	60	0.741
African-American	8	10	5	1.000
Asian	8	5	10	1.000
Other	19	15	25	0.697
BMI (kg/m^2)	28.20 ± 5.58	29.57 ± 5.54	26.22 ± 5.26	0.128
LVEF (%)	52.05 ± 10.74	50.75 ± 12.28	53.42 ± 8.98	0.445
Prior unsuccessful ablations (mean n/patient)	0.45 ± 0.80	0.55 ± 0.94	0.33 ± 0.77	0.409
Antiarrhythmic drugs (mean n/patient)	0.32 ± 0.70	0.30 ± 0.66	0.33 ± 0.77	0.886
PVC Burden (mean n/24-h Holter)	24,560.71 ± 19,047.97	29,429.76 ± 19,107.61	12,875 ± 14,333.73	0.104
Clinical arrhythmia				
Frequent PVCs	20	11	9	0.752
NSVT	15	6	9	0.466
Sustained VT	5	3	2	1.000
VT CL (ms)	414 ± 118	421 ± 97	359 ± 143	0.822
Pre-QRS activation time (ms)	−33.90 ± 9.17	−36.47 ± 9.26	−27.50 ± 5.24	0.039

BMI = body mass index; CL = cycle length; LVEF = left ventricular ejection fraction; LVOT = left ventricular outflow tract; NSVT = nonsustained ventricular tachycardia; PVC = premature ventricular contraction; RVOT = right ventricular outflow tract; VT = ventricular tachycardia.

and incomplete records (n = 99), we identified 40 cases of successful OTVT ablation with lead-V_3 transition that met our inclusion criteria. Patient demographic data between RVOT and LVOT groups were similar at baseline (Table 1). The RVOT cohort consisted of 80% anteroseptal sites and 20% posteroseptal sites. The LVOT cohort consisted of 35% LCC, 15% RCC, 40% left-right coronary cusp junction, and 10% great cardiac vein near the anterior interventricular vein.

Absolute measurements of PVC and SR QRS indexes are listed in Table 2. The R-wave amplitude was greater for LVOT compared with RVOT PVCs in leads V_2 ($p < 0.001$) and V_3 ($p < 0.001$). The R-wave duration ratio of the PVC to SR in lead V_2 ($p = 0.002$) and lead V_3 ($p = 0.026$) was also significantly greater for LVOT compared with RVOT PVCs. The V_2 transition ratio was significantly greater for LVOT (range 0.42 to 2.89) compared with RVOT origin (range 0.02 to 0.57) ($p < 0.001$); however, the V_3 transition ratio was not significantly different between PVCs of LVOT versus RVOT origin ($p = 0.093$) (Fig. 4). The overall QRS duration was longer for LVOT compared with RVOT PVCs ($p = 0.048$).

In a multivariate logistic regression, including R-wave duration in leads V_1 and V_2, R-wave amplitude in leads V_2 and V_3, and the R-wave transition ratio in lead V_2, the V_2 R-wave transition ratio was the only independent predictor of PVC origin ($p < 0.001$, 95% confidence interval: 0.01 to 0.41).

A receiver operating characteristic curve for the retrospective data is illustrated in Figure 5. A V_2 transition ratio ≥0.6 predicted an LVOT origin with a sensitivity of 95% and specificity of 100% (Fig. 4). This cutoff yielded a positive predictive value of 100% and a negative predictive value of 95%. A more practical cutoff of ≥0.5 yields a sensitivity of 95% and specificity of 95%.

The simple qualitative measure of PVC precordial transition (R>S) occurring at or before the SR transition (R>S) had a sensitivity of 47% and specificity of 64% for identifying an LVOT origin. However, a PVC precordial transition occurring later than the SR transition had a 19% sensitivity and 100% specificity for RVOT origin. Therefore, a PVC that transitions later than SR effectively rules out an LVOT origin. The surface ECGs of representative PVCs with lead V_3 R/S transition are illustrated adjacent to their corresponding sinus beats in Figure 6.

Prospective analysis. The ECG measurements of the ensuing 21 cases of OTVT who underwent successful RF ablation at our institution were performed. The algorithm was able to correctly predict the site of successful ablation (LVOT vs. RVOT) in 91% (19 of 21) of cases. The interobserver agreement in measurement of R- and S-wave amplitudes yielded an intraclass correlation coefficient of 0.966 (95% confidence interval: 0.937 to 0.982; $p < 0.001$), and the kappa statistic for classification agreement was 0.877 ($p < 0.001$). With the simple qualitative measure alone, where LVOT origin is suggested by a PVC transition at or earlier than the SR transition, there was 71% accuracy in diagnosing the PVC origin (100% interobserver agreement). On the other hand, when the PVC transition occurred later than the sinus rhythm transition, an LVOT origin could be excluded with 100% accuracy. A proposed algorithm combining the qualitative and quantitative measures is shown in Figure 7.

Discussion

We present a novel surface ECG measurement, the V_2 transition ratio, for distinguishing LVOT from RVOT origin in patients with left bundle branch pattern idiopathic

JACC Vol. 57, No. 22, 2011
May 31, 2011:2255–62

Table 2 Electrocardiographic Measurements and Sites of Successful Ablation in the Retrospective Cohort

	LVOT	RVOT	p Value
Lead I			
R amplitude PVC (mV)	0.30 ± 0.26	0.21 ± 0.25	0.322
S amplitude PVC (mV)	0.17 ± 0.19	0.23 ± 0.25	0.295
R amplitude SR (mV)	0.59 ± 0.39	0.56 ± 0.29	0.793
S amplitude SR (mV)	0.11 ± 0.11	0.13 ± 0.09	0.522
R amplitude ratio	0.52 ± 0.50	0.35 ± 0.31	0.287
Lead II			
R amplitude PVC (mV)	1.84 ± 0.32	1.66 ± 0.52	0.246
S amplitude PVC (mV)	0.24 ± 0.11	0.32 ± 0.16	0.098
R amplitude SR (mV)	0.78 ± 0.43	0.76 ± 0.25	0.867
S amplitude SR	0.08 ± 0.11	0.12 ± 0.08	0.332
R amplitude ratio	2.90 ± 1.52	2.27 ± 0.6	0.154
Lead III			
R amplitude PVC (mV)	1.71 ± 0.51	1.70 ± 0.68	0.965
S amplitude PVC (mV)	0.24 ± 0.12	0.39 ± 0.12	0.018
R amplitude SR (mV)	0.46 ± 0.38	0.42 ± 0.31	0.744
S amplitude SR (mV)	0.16 ± 0.24	0.14 ± 0.19	0.833
R amplitude ratio	8.17 ± 11.05	6.94 ± 6.48	0.713
Lead aVF			
R amplitude PVC (mV)	1.76 ± 0.40	1.68 ± 0.60	0.642
S amplitude PVC (mV)	0.24 ± 0.11	0.34 ± 0.16	0.043
R amplitude SR (mV)	0.56 ± 0.41	0.73 ± 0.71	0.387
S amplitude SR (mV)	0.09 ± 0.12	0.10 ± 0.08	0.847
R amplitude ratio	4.22 ± 3.63	5.30 ± 6.08	0.553
Lead V_1			
R duration PVC (ms)	62.50 ± 17.27	62.20 ± 37.06	0.978
R duration SR (ms)	25.26 ± 8.24	39.71 ± 25.63	0.028
R duration ratio	2.65 ± 0.91	2.12 ± 1.83	0.336
Lead V_2			
R amplitude PVC (mV)	0.49 ± 0.30	0.18 ± 0.15	<0.001
S amplitude PVC (mV)	1.13 ± 0.47	1.93 ± 0.72	<0.001
R amplitude SR (mV)	0.37 ± 0.20	0.63 ± 0.54	0.054
S amplitude SR (mV)	1.00 ± 0.30	0.79 ± 0.42	0.071
R amplitude ratio	1.65 ± 1.13	0.41 ± 0.30	<0.001
R duration PVC (ms)	63.61 ± 19.32	53.47 ± 17.26	0.125
R duration SR (ms)	31.58 ± 9.84	39.87 ± 14.44	0.055
R duration ratio	2.08 ± 0.57	1.42 ± 0.54	0.002
V_2 transition ratio	1.27 ± 0.60	0.23 ± 0.16	<0.001
Lead V_3			
R amplitude PVC (mV)	1.44 ± 0.44	0.95 ± 0.39	0.001
S amplitude PVC (mV)	0.32 ± 0.32	0.35 ± 0.26	0.751
R amplitude SR (mV)	0.85 ± 0.66	0.78 ± 0.51	0.717
S amplitude SR (mV)	1.00 ± 0.30	0.79 ± 0.42	0.071
R amplitude ratio	3.26 ± 3.32	1.90 ± 1.45	0.101
R duration PVC (ms)	103.74 ± 25.16	92.67 ± 22.19	0.190
R duration SR (ms)	39.63 ± 16.00	46.00 ± 14.44	0.238
R duration ratio	2.92 ± 1.011	2.18 ± 0.78	0.026
V_3 transition ratio	2.70 ± 2.68	1.60 ± 1.02	0.093
Other parameters			
QRS duration PVC (ms)	155.89 ± 22.34	142.07 ± 13.25	0.048
QRS axis PVC (°)	87.19 ± 24.01	91.07 ± 18.10	0.625

SR = sinus rhythm; other abbreviations as in Table 1.

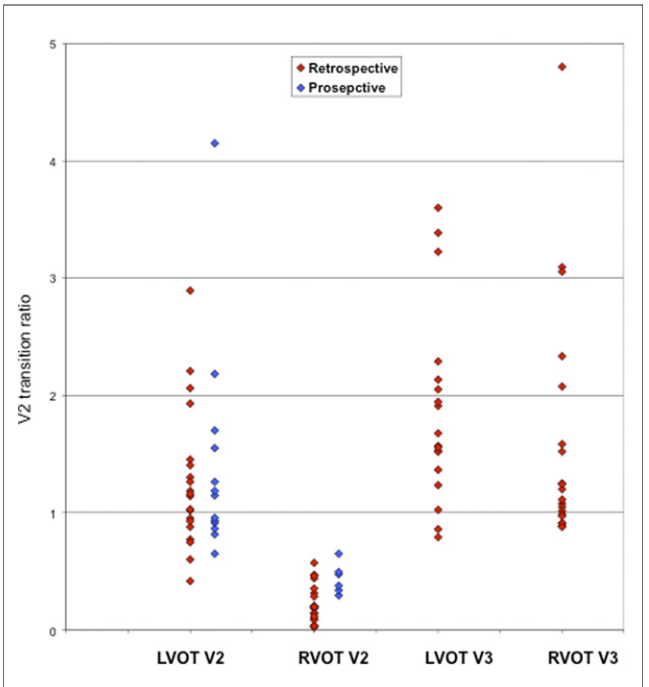

Figure 4 Scatterplot Illustrating Calculated V_2 and V_3 Transition Ratios

The V_2 transition ratio distinguished right ventricular outflow tract (RVOT) from left ventricular outflow tract (LVOT) site of origin in the retrospective **(red)** and prospective **(blue)** cohorts; RVOT sites had a significantly lower V_2 transition ratio ($p < 0.001$). The V_3 transition ratio did not distinguish RVOT from LVOT site of origin.

VT/PVCs with lead V_3 precordial transition. This measure accounts for variations in body habitus, cardiac rotation, respiratory variation, and ECG lead positioning by measuring precordial transition during the PVC/VT relative to the

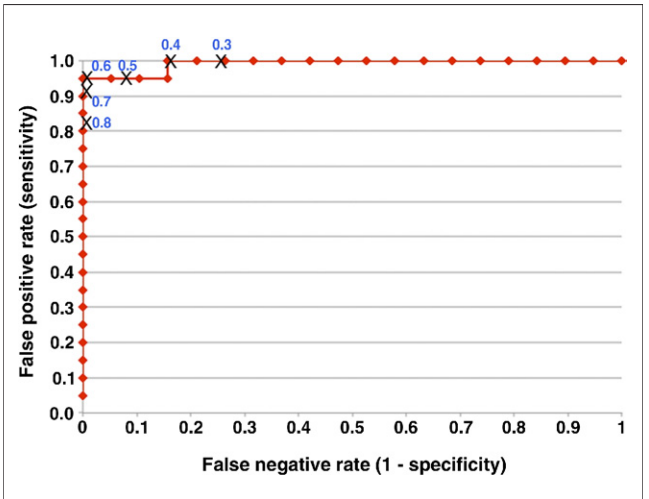

Figure 5 Receiver-Operating Characteristic Curve for the V_2 Transition Ratios in the Retrospective Cohort

Sensitivity and specificity for predicting left ventricular outflow tract premature ventricular contraction/ventricular tachycardia origin are indicated for different V_2 transition ratio cutoffs. Area under the curve = 0.992.

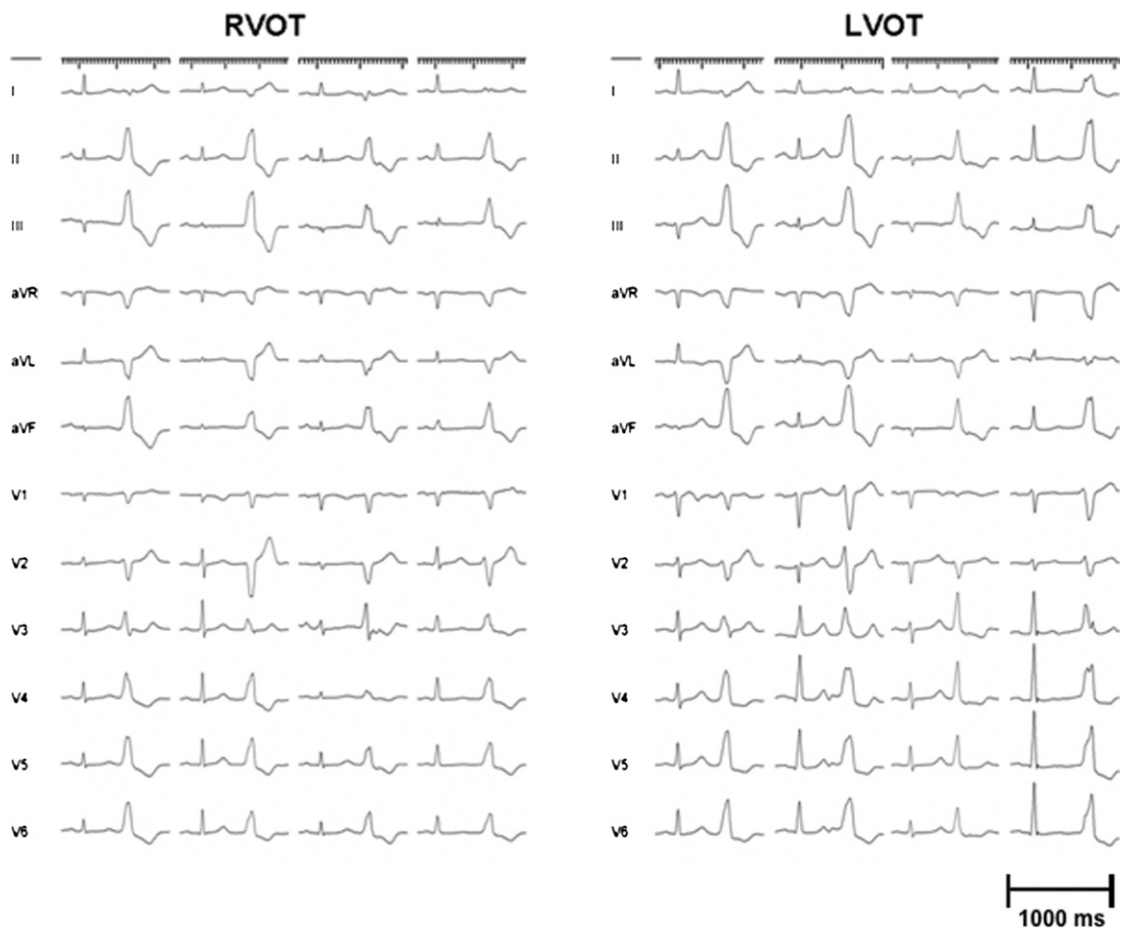

Figure 6 **Representative Outflow Tract PVC Surface ECG Recordings**

The SR QRS and PVC morphologies from the retrospective cohort demonstrating the relatively earlier SR precordial transition in RVOT **(left)** compared with patients with LVOT PVC origin **(right)**. The ECG speed: 25 mm/s. Lead gain standardized to 1/16. Abbreviations as in Figures 3 and 4.

SR precordial transition. A V_2 transition ratio ≥0.6 predicted an LVOT origin with 95% sensitivity and 100% specificity. For patients referred for catheter ablation of OTVT, this simple ECG measurement might be performed in the office both to help plan an ablation strategy and to enhance patient counseling with regard to procedural time, potential outcome, and risks associated with arterial access, mapping, and ablation.

One might argue that the V_2 transition ratio is cumbersome for everyday use in clinical practice. In the electrophysiology lab, this measurement is easily made with digital calipers available on any clinical electrophysiologic recording system. For more practical clinical use, however, we also found that a precordial transition during the PVC/VT that occurs later than the SR transition excludes an LVOT origin with 100% accuracy. This simple measure can be easily used by any cardiologist or electrophysiologist when counseling patients about PVC ablation.

Prior work. There have been many prior studies evaluating the surface ECG for localization of OTVTs. Dixit et al. (12) in our laboratory established the typical ECG features for RVOT septal and free wall PVC origin. Ouyang et al. (13) found that a greater R-wave duration and R/S-wave amplitude ratio in leads V_1 or V_2 reliably predicted an aortic sinus cusp compared with RVOT origin. Lin et al. (11) described site-specific ECG features of PVCs originating from the aortic cusps with electroanatomic and ICE-guided pacemapping. They concluded that the LCC typically produces a precordial transition by V_2, whereas the RCC demonstrated a precordial transition by lead V_3. Furthermore, studies by Yamada et al. (9) and Bala et al. (10) characterized the left-right coronary cusp junction as often possessing QRS in leads V_1 to V_3 or QS in lead V_1. One previous study of patients undergoing RF catheter ablation for idiopathic VT or symptomatic PVCs implicated the R-wave amplitude proportion as a means of discriminating between RVOT and extra-RVOT foci; however, the study was limited by small size, lack of prospective evaluation, or an assessment of the frontal plane (17). It has previously been shown that lead V_3 R/S transition is highly variable,

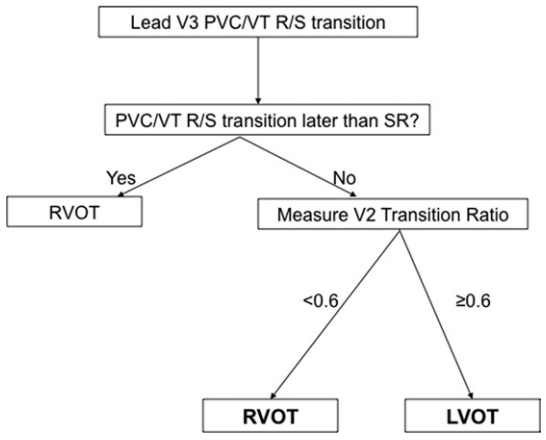

Figure 7 **Diagnostic Algorithm for Outflow Tract VT With Lead V_3 PVC/VT R/S Transition**

If the PVC/ventricular tachycardia (VT) transition to an R>S occurs later than the SR transition (i.e., SR transition lead V_1 or V_2), then the PVC origin is the RVOT (100% specificity). If the PVC transition occurs at or earlier than the SR transition (i.e., SR transition lead V_3 or later), then the V_2 transition ratio is measured. If the transition ratio is <0.6, then RVOT origin is likely. If the transition ratio is ≥0.6, then LVOT origin is likely (sensitivity 95%, specificity 100%). Abbreviations as in Figures 3 and 4.

with only 55% sensitivity and 38% specificity for predicting an RVOT origin. (16).

Study limitations. The sample size, although comparable to other studies of OTVT, is small and should be validated further in larger populations. We used the location of the ablation catheter in the RVOT or LVOT during abolition of the clinical arrhythmia as the PVC/VT site of origin. It is possible that ablation lesions from the RVOT or LVOT extended into adjacent structures and that a PVC with epicardial or LVOT origin could be abolished from the RVOT. This might explain the few cases that did not match our criteria. Detailed measurements with digital calipers were used to develop the algorithm. It is possible that such measurements will prove more difficult in clinical practice, although as stated in the preceding text, the theory of incorporating the SR precordial transition into the decision algorithm for predicting a site of PVC origin still applies.

Conclusions

We present a novel electrocardiographic measure of the ratio of the VT and SR precordial transition, "the V_2 transition ratio," which can reliably distinguish left from right outflow tract PVC/VT origin in patients with OTVT and lead V_3 precordial R/S transition. A V_2 transition ratio ≥0.6 predicts an LVOT origin with high sensitivity and specificity. A precordial transition later than the SR transition excludes an LVOT VT origin. This algorithm might lead to improved patient counseling and planning of medical therapy or ablation procedures in patients referred for ablation of outflow tract tachycardias.

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